



D Save Workspace
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Active

- L1: (5286) hepatitis adj b
- L2: (973700) polymerase or pol or p
- L3: (878095) mutant or mutation or variant or v
- L4: (11838) 2 with 3
- L5: (567) 1 and 4
- L6: (22) 1 same 4
- L7: (23) hbv same 4
- L8: (13) 7 not 6
- L9: (3063) hepatitis adj b or hbv
- L10: (2256822) polymerase or pol or p
- L11: (287274) mutant or mutation or variant or v
- L12: (22) 9 and (10 with 11)

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Databases

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USPAT

Patents

Synonyms

Default operator: OR

 Highlight all hit terms initially

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BRS f...

IS&R

Image

Text

Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error
1	BRS L1	5286	hepatitis adj b	USPAT	2001/03/28 15:40		
2	BRS L2	973700	polymerase or pol or p	USPAT	2001/03/28 15:40		
3	BRS L3	878095	mutant or mutation or variant or variation or mutat\$	USPAT	2001/03/28 15:41		
4	BRS L4	11838	2 with 3	USPAT	2001/03/28 15:24		
5	BRS L5	567	1 and 4	USPAT	2001/03/28 15:24		
6	BRS L6	22	1 same 4	USPAT	2001/03/28 15:35		
7	BRS L7	23	hbv same 4	USPAT	2001/03/28 15:35		
8	BRS L8	13	7 not 6	USPAT	2001/03/28 15:35		
9	BRS L9	3063	hepatitis adj b or hbv	EPO, JPO, DERWENT	2001/03/28 15:40		
10	BRS L10	2256822	polymerase or pol or p	EPO, JPO, DERWENT	2001/03/28 15:41		
11	BRS L11	287274	mutant or mutation or variant or variation or mutat\$	EPO, JPO, DERWENT	2001/03/28 15:41		
12	BRS L12	22	9 and (10 with 11)	EPO, JPO, DERWENT	2001/03/28 15:41		



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Active

- L1: (5286) hepatitis adj b
- L2: (973700) polymerase or pol or p
- L3: (878095) mutant or mutation or variant or v
- L4: (11838) 2 with 3
- L5: (567) 1 and 4
- L6: (22) 1 same 4
- L7: (23) hbv same 4
- L8: (13) 7 not 6
- L9: (3063) hepatitis adj b or hbv
- L10: (2256822) polymerase or pol or p
- L11: (287274) mutant or mutation or variant or v
- L12: (22) 9 and (10 with 11)

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DBs: USPAT
Default operator: OR

Plurals Synonyms
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BRS IS&P ... Image Text

	Type	L #	Hits	Search Text	DBs	Time Stamp	Comments	Error
1	BRS	L1	5286	hepatitis adj b	USPAT	2001/03/28 15:40		
2	BRS	L2	973700	polymerase or pol or p	USPAT	2001/03/28 15:40		
3	BRS	L3	878095	mutant or mutation or variant or variation or mutat\$	USPAT	2001/03/28 15:41		
4	BRS	L4	11838	2 with 3	USPAT	2001/03/28 15:24		
5	BRS	L5	567	1 and 4	USPAT	2001/03/28 15:24		
6	BRS	L6	22	1 same 4	USPAT	2001/03/28 15:35		
7	BRS	L7	23	hbv same 4	USPAT	2001/03/28 15:35		
8	BRS	L8	13	7 not 6	USPAT	2001/03/28 15:35		
9	BRS	L9	3063	hepatitis adj b or hbv	EPO; JPO; DERWENT	2001/03/28 15:40		
10	BRS	L10	225682 2	polymerase or pol or p	EPO; JPO; DERWENT	2001/03/28 15:41		
11	BRS	L11	287274	mutant or mutation or variant or variation or mutat\$	EPO; JPO; DERWENT	2001/03/28 15:41		
12	BRS	L12	22	9 and (10 with 11)	EPO; JPO; DERWENT	2001/03/28 15:41		

? b 155
28mar01 10:04:12 User208669 Session D1812.1
\$0.22 0.061 DialUnits File1
\$0.22 Estimated cost File1
\$0.22 Estimated cost this search
\$0.22 Estimated total session cost 0.061 DialUnits

File 155: MEDLINE(R) 1966-2000/Dec W4
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to resume in March 2001. For other NLM information see Help News155.

Set Items Description
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Set	Items	Description
S1	69	DUCK AND HEPATITIS AND POLYMERASE AND SEQUENC?
S2	17	POLYMERASE (3N) SEQUENC? AND S1
S3	3	AMINO AND S2

? ts27/2 11 15 17

27/2
DIALOG(R)File 155: MEDLINE(R)
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09196169 97368434

Sequence heterogeneity of heron hepatitis B virus genomes determined by full-length DNA amplification and direct sequencing reveals novel and unique features.

Netter HJ; Chassot S; Chang SF; Cova L; Will H
Heinrich-Pette-Institut fur experimentelle Virologie und Immunologie,
Universitat Hamburg, Germany.

Journal of general virology (ENGLAND) Jul 1997, 78 (Pt 7) p1707-18,
ISSN 0022-1317 Journal Code: 19B

Languages: ENGLISH

Document type: JOURNAL ARTICLE

So far, only a single heron hepatitis B virus genome (HHBV-4) has been cloned and sequenced. Therefore, neither the significance of its sequence divergence from other avian hepadnaviruses nor the sequence variability of HHBV genomes in general are known. Here we have analysed the sequence heterogeneity of HHBV genome populations in several sera from naturally infected herons. A highly sensitive PCR method for full-length HHBV genome amplification was established which allowed direct sequencing of entire HHBV populations without prior cloning. Sequences of HHBV genomes from four sera were thus obtained which differed from those of HHBV-4 by up to 7%. Some of the divergent nucleotides and the corresponding amino acids of the predicted viral proteins were conserved in all four new HHBV isolates and varied only in HHBV-4. This indicates that the HHBV-4 genome is not in all

aspects representative of this class of viruses. Interestingly, a highly conserved ORF upstream of the C-gene present in a position analogous to that of the mammalian hepadnavirus X-gene became apparent in all HHBV genomes. In contrast to the duck hepadnaviruses, the small (sAg-S) instead of the largest (sAg-L) envelope protein of all HHBVs has a myristylation site. These data confirm the significant sequence divergence of HHBV from other avian hepadnaviruses. Moreover, they show that HHBV has low sequence variability and indicate two new and unique features not evident in other avihepadnaviruses: an additional, highly conserved gene and potential myristylation of the sAg-S instead of the sAg-L envelope protein.

27/1

DIALOG(R)File 155: MEDLINE(R)

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08159805 94202286

Hepadnavirus P protein utilizes a tyrosine residue in the TP domain to prime reverse transcription.

Weber M; Bronsema V; Bartos H; Bosserhoff A; Bartenschlager R; Schaller H
ZMBH, University of Heidelberg, Germany.
Journal of virology (UNITED STATES) May 1994, 68 (5) p2994-9, ISSN
0022-538X Journal Code: KCV

Languages: ENGLISH

Document type: JOURNAL ARTICLE

Hepadnavirus DNA minus strands are covalently linked at their 5' terminus to the viral P gene product, which has been taken to indicate that the hepadnaviral polymerase polypeptide itself also functions as a protein primer for initiating reverse transcription of the RNA pregenome. The present study confirms this indication by identifying the nucleotide-linked amino acid in the P protein sequence of the duck hepatitis B virus (DHBV).

In a first set of experiments, mutational analysis of three phylogenetically conserved tyrosine residues in the DNA terminal (TP) domain indicated that of these, only tyrosine 96 was essential for both viral DNA synthesis in transfected cells and priming of DNA synthesis in a cell-free system. This assignment was confirmed by direct biochemical analysis: tryptic peptides from the DHBV P protein, 32P labelled at the priming amino acid by the initiating dGTP and additionally labelled internally by [³⁵S]methionine, were isolated and analyzed in parallel to reference peptides synthesized chemically and 33P labelled by a tyrosine kinase. Mobility in high-performance liquid chromatography, as well as the release in stepwise amino acid sequencing of phospholabel and of [³⁵S]methionine, identified the priming amino acid unequivocally as the tyrosine in the sequence 91KLSGLYQMKG99, which is located in the center of the TP domain. Conserved sequence motifs surrounding Ty-96 allow the prediction of the priming tyrosine in other hepadnaviruses. Weak sequence similarity to picornavirus genome-linked polypeptides (VPgs) and similar gene organization suggest a common origin for the mechanisms that use protein priming to initiate synthesis of viral DNA genomes or RNA genomes.

from an RNA template.

2/7/15 DIALOG(R)File 155.MEDLINE(R)
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07770945 93252186

Inflammation of the liver causes mutations in duck hepatitis B virus genome.

Fukuda R; Kohge N; Akagi S; Xuan NT; Tokuda A; Fukumoto S
 Second Department of Internal Medicine, Shimane Medical University,
 Izumo, Japan.

Gastroenterologia Japonica (JAPAN) Apr 1993, 28 (2) p254-8, ISSN
 0435-1339 Journal Code: FHJY

Languages: ENGLISH

Document type: JOURNAL ARTICLE

To investigate whether hepatitis causes mutation in the viral genome, DNA sequences in the pre-core region of duck hepatitis B virus (DHBV) DNA were analyzed in both ducks with hepatitis and without hepatitis. Five DHBV carrier ducks were injected with DHBV particle proteins purified from duck serum with Freund's complete adjuvant (FCA) intraperitoneally from 14 day postpatch for 9 weeks (immunized group). Serum was drawn at the end of the 1st and 4th week after the 1st injection of DHBV particle protein and ducks were killed at the end of the 9th week to obtain the liver. Another five ducks without treatment were used as controls. All ducks of the immunized group showed moderate to severe hepatitis at the 9th week. All ducks in the immunized group showed one mutation except one duck that showed two mutations only at the 9th week. Mutations were observed in the 5th, 13th, 21st, 22nd, and 28th codon of the pre-core region. All of them were point mutation at the 3rd base in the triplets. The frequency of mutation was different in each duck from 20% to 60% but not 100%. There was no mutations in ducks in control group. These results suggest that hepatitis causes mutation in the pre-core lesion genome of duck hepatitis B virus.

2/7/17 DIALOG(R)File 155.MEDLINE(R)
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 04532573 81182544

Genetic variation among hepatitis B and related viruses.
 Robinson WS

Annals of the New York Academy of Sciences (UNITED STATES) 1980, 354 p371-8, ISSN 0077-8923 Journal Code: 5NM
 Contract/Grant No.: AI-13526, AI, NIADDK

Languages: ENGLISH

Document type: JOURNAL ARTICLE, REVIEW
 Hepatitis B virus (HBV) of man has several characteristics that distinguish it from viruses of other groups. These include its ultrastructure, viral DNA size and structure, a virion DNA polymerase which

repairs a single-stranded region in the viral DNA, liver tropism, character of persistent infection, and association with hepatitis and hepatocellular carcinoma. Recently three other viruses have been found in other animal species that appear to share these characteristics although the viruses are not identical. HBV, Woodchuck hepatitis virus (WHV), ground squirrel hepatitis virus (GSHV), and duck hepatitis virus (DHV) appear to be members of a new virus group that might be designated the Herpadna virus group. Genetic variation among hepatitis B viruses includes the antigenic variation in the surface antigen (HBsAg) which constitutes the known HBsAg subtypes. There is also frequent variation in DNA base sequence among HBVs isolated from different patients. (38 Refs.)

? s duck and hepatitis and (p or pol) (3n) sequenc?
 ? s duck and hepatitis and (p or pol) (3n) sequenc?

2929 DUCK
 92857 HEPATITIS
 1792618 P
 5086 POL
 530876 SEQUENC?
 3591 (P OR POL)(3N)SEQUENC?
 S4 3 DUCK AND HEPATITIS AND (P OR POL) (3N) SEQUENC?
 ? t s4/7/3

4/7/3
 DIALOG(R)File 155.MEDLINE(R)
 (c) format only 2000 Dialog Corporation. All rts. reserv.
 06956836 90311362
 Mechanism of translation of the hepadnaviral polymerase (P) gene.
 Chang LJ; Ganem D; Varmus HE
 Department of Microbiology and Immunology, University of California, San Francisco 94143.
 Proceedings of the National Academy of Sciences of the United States of America (UNITED STATES) Jul 1990, 87 (13) p5158-62, ISSN 0027-8424
 Journal Code: PV3

Languages: ENGLISH

Document type: JOURNAL ARTICLE

Unlike many other reverse transcriptase genes, the polymerase (P) gene of the hepatitis B viruses is expressed by translational initiation from its own AUG codon rather than by ribosomal frameshifting during translation of the overlapping core gene (C). To explore the mechanism of its translation, we have fused the P gene of duck hepatitis B virus to the bacterial lacZ gene at a point 3' to the C-P overlap; this allows detection of the products of P translation with antisera to the lacZ-encoded protein. The C and P/Z coding regions were cloned downstream of a heterologous promoter and expressed in COS-7 cells. A single, bicistronic mRNA containing both C and P sequences is detected in these cells, and translational initiation occurs efficiently at the internally situated P AUG. Mutations affecting C translation have only minimal effects on P expression, in contrast to what would be expected from a modified scanning model for translation. We

conclude that P translation depends on a mechanism other than scanning to allow internal entry of ribosomes to the region of the P initiator.
 ? s duck and hepatitis and (p or pol or polymerase) (8n) sequence?

2929 DUCK

92857 HEPATITIS

1792618 P

5086 POL

152173 POLYMERASE

530876 SEQUENC?

56838 ((P OR POL) OR POLYMERASE)(8N)SEQUENC?

SS 36 DUCK AND HEPATITIS AND (P OR POL OR POLYMERASE) (8N) SEQUENC?

? t s5/7/36

5/7/36
 DIALOG(R)File 155:MEDLINE(R)

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 03993100 84138772

Nucleotide sequence of a cloned duck hepatitis B virus genome: comparison with woodchuck and human hepatitis B virus sequences.

Mandart E; Kay A; Galibert F

Journal of virology (UNITED STATES) Mar 1984, 49 (3) p782-92, ISSN 0022-538X Journal Code: KCV

Languages: ENGLISH

Document type: JOURNAL ARTICLE

The nucleotide sequence of an EcoRI duck hepatitis B virus (DHBV) clone was elucidated by using the Maxam and Gilbert method. This sequence, which is 3,021 nucleotides long, was compared with the two previously analyzed hepatitis B-like viruses (human and woodchuck). From this comparison, it was shown that DHBV is derived from an ancestor common to the two others but has a slightly different genomic organization. There was no intergenic region between genes 5 and 8, which were fused into a single open reading frame in DHBV. Genes for the surface and core proteins were assigned to open reading frames 7 and 5/8. Amino acid comparisons showed some structural relationship between gene 6 product and avian reverse transcriptase, suggesting either evolution from a common ancestor or convergence to some particular structure to fulfill a specific function.

This should be correlated with the synthesis of an RNA intermediate during DNA replication. This is also taken as an argument in favor of the hypothesis that gene 6 codes for the DNA polymerase that is found within the virion. DNA sequence comparison also showed that the two mammalian hepatitis B viruses are more homologous to each other than they are to DHBV, indicating that DHBV starts to evolve on its own earlier than the two other viruses, as do birds compared with mammals. From this it is proposed that the viruses evolved in a fashion parallel to the species they infect.

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28mar01 11:06:08 User208669 Session D1813.1

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\$0.22 Estimated cost this search

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3/7/11

DIALOG(R)File 5:Biosis Previews(R)

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09716016 BIOSIS NO.: 199598170934

Molecular analysis of the essential and nonessential genetic elements in the genome of peanut chlorotic streak caulimovirus.

AUTHOR: Mushegian A.R.(a); Wolff J.A.; Richins R.D.; Shepherd R.J.

AUTHOR ADDRESS: (a)Dep. Microbiol., SC-42, Univ. Washington, Seattle, WA 98195**USA

JOURNAL: Virology 206 (2):p823-834 1995

ISSN: 0042-6822

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: The DNA genome of caulimoviruses contains a set of essential genes: I (movement gene), IV (major capsid protein gene), V (reverse transcriptase gene), and VI (gene coding for a post-transcriptional activator of the expression of other virus genes). In peanut chlorotic streak caulimovirus (PCISV), three ORFs, A, B, and C, are located between genes I and IV. They are dissimilar to other caulimovirus ORFs. ORF VII of PCISV is a homolog of ORF VII of soybean chlorotic mottle caulimovirus (SoCMV), but is not similar to the nonconserved ORF VII in other caulimoviruses. The sequence complementary to a portion of RNA-Met, thought to be essential for the priming of minus-strand DNA synthesis in caulimoviruses, is located within the coding sequence of ORF A. To explore the functional significance of ORFs VII, A, B, and C, various mutations were engineered into an infectious DNA clone of PCISV. ORFs VII and B are shown to be dispensable, while ORFs A and C are essential. ORF C is a possible functional equivalent of gene III in other caulimoviruses. Sequences within ORF A that are required for efficient priming of minus-strand synthesis are likely to extend beyond the 12-bp RNA-binding site. Complete deletion of ORF VII was correlated with severe symptoms, notably with the necrosis of apical meristems. Significance of these observations for the understanding of replication and pathogenesis of plant pararetroviruses and for the improvement of caulimovirus-based expression vectors is discussed.

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DIALOG(R)File 5:Biosis Previews(R)

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07818061 BIOSIS NO.: 000092099247

A NATURALLY OCCURRING DELETION MUTANT OF FIGWORT MOSAIC VIRUS CAULIMOVI

IS GENERATED BY RNA SPLICING

AUTHOR: SCHOLTHOF H B; WU F C; RICHINS R D; SHEPHERD R J

AUTHOR ADDRESS: DEP. PLANT PATHOLOGY, UNIVERSITY KENTUCKY, LEXINGTON, KY. 40546-0091.

JOURNAL: VIROLOGY 184 (1). 1991. 290-298. 1991

FULL JOURNAL NAME: Virology

CODEN: VIRLA

RECORD TYPE: Abstract

LANGUAGE: ENGLISH

ABSTRACT: A naturally occurring deletion mutant is observed in plants infected with figwort mosaic virus (FMV), a caulimovirus. The encapsidated mutant genome is formed spontaneously in association with two different strains of FMV in four host plant species. The mutant also appears when cloned wild-type viral DNA is used as the inoculum. The deletion mutant alone is not infectious and it appears unable to replicate after its formation, even in the presence of wild-type virus. The gene for chloramphenicol acetyltransferase was inserted at different positions in the deletion mutant genome, and subsequent transient assays showed that gene expression of the mutant occurs despite the deletion. Sequence analyses of the mutant genome revealed a deletion of a 1237-bp segment encompassing a major portion of the coat protein gene and the 5' end of the downstream reverse transcriptase gene. This deletion is associated with consensus signals for RNA splicing including the conserved 5' and 3' splice sites plus surrounding sequences, putative branch point(s) for lariat formation, and an extremely high adenosine content (41%) of the removed fragment. This suggests that splicing of the FMV full-length transcript has occurred prior to reverse transcription and this accounts for the presence and accumulation of encapsidated DNAs with the same deletion.

3/7/19 DIALOG(R)File 5:Biota Previews(R)
 (c) 2001 BIOSIS. All rts. reserv.
 07247995 BIOSIS NO.: 000090027871
OPEN READING FRAME VIII IS NOT REQUIRED FOR VIABILITY OF CAULIFLOWER MOSAIC VIRUS
 AUTHOR: SCHULTZE M; JIRICNY J; HOHN T
 AUTHOR ADDRESS: FRIEDRICH MIESCHER-INST., P.O. BOX 2543, CH-4002 BASEL, SWITZ.

JOURNAL: VIROLOGY 176 (2). 1990. 662-664. 1990
 FULL JOURNAL NAME: Virology
 CODEN: VIRLA
 RECORD TYPE: Abstract
 LANGUAGE: ENGLISH

ABSTRACT: Open reading frame (ORF) VIII of cauliflower mosaic virus (CaMV) was analyzed by site-directed mutagenesis in order to investigate its potential function for the viral life cycle. Removal of either the start or the stop codon of ORF VIII, as well as interruption of ORF VIII by a new stop codon, did not affect infectivity. Unlike certain ORF VII mutants all three ORF VIII mutants are stable. Hence the ORF VIII product is not essential and ORF VIII mutations do not have deleterious polar effects on the expression of the downstream ORF V, which codes for the viral protease/reverse transcriptase.

3/7/20 DIALOG(R)File 5:Biota Previews(R)
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 07055885 BIOSIS NO.: 000089125988
THE REVERSE TRANSCRIPTASE GENE OF CAULIFLOWER MOSAIC VIRUS IS TRANSLATED SEPARATELY FROM THE CAPSID GENE
 AUTHOR: SCHULTZE M; HOHN T; JIRICNY J
 AUTHOR ADDRESS: FRIEDRICH MIESCHER-INST., P.O. BOX 2543, CH-4002 BASEL,
 SWITZ.
 JOURNAL: EMBO (EUR MOL BIOL ORGAN) J 9 (4). 1990. 1177-1186. 1990
 FULL JOURNAL NAME: EMBO (European Molecular Biology Organization) Journal
 CODEN: EMIOD
 RECORD TYPE: Abstract
 LANGUAGE: ENGLISH
ABSTRACT: Cauliflower mosaic virus (CaMV) possesses start codons at the beginning of its reverse transcriptase (RT) gene (ORF V) suggesting that, unlike in retroviruses and retrotransposons, it is translated independently from the capsid gene (ORF-IV). To test this hypothesis a mutational analysis of the CaMV ORF IV/V overlapping region was performed. Mutants in which both ORFs are separated by stop codons in all three reading frames are viable and stable, while mutations affecting the first two AUG codons of ORF V are either lethal or unstable, giving rise to true and second site reversions. Mutants in which the AUG codons were replaced by ACG or AAG reverted only slowly and ACC could direct the synthesis of small amounts of reporter enzyme in transfected plant protoplasts, showing that this codon can act in plant cells as a weak start codon. CaMV has apparently developed a strategy for translation of the RT gene different from that in retroviruses and retrotransposons, but similar to that of hepadnaviruses, another group of pararetroviruses. The separate translation of the RT gene as a common feature of pararetroviruses might reflect the difference in their life cycle in comparison with retroviruses.

3/7/21 DIALOG(R)File 5:Biota Previews(R)

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 06147396 BIOSIS NO.: 000085110548
 A VIABLE MUTATION IN CAULIFLOWER MOSAIC VIRUS A
 RETROVIRUS-LIKE PLANT VIRUS
 SEPARATES ITS CAPSID PROTEIN AND POLYMERASE GENES
 AUTHOR: PENSWICK J; HUBLER R; HOHN T
 AUTHOR ADDRESS: FRIEDRICH-MESCHER-INST., P.O. BOX 2543, CH-4002
 BASEL,
 SWITZERLAND.
 JOURNAL: J VIROL 62 (4). 1988. 1460-1463. 1988
 FULL JOURNAL NAME: Journal of Virology
 CODEN: JOVI
 RECORD TYPE: Abstract
 LANGUAGE: ENGLISH
 ABSTRACT: A viable strain of cauliflower mosaic virus is described which arose by illegitimate recombination of two lethal parents. In this strain, the normally overlapping open reading frames IV and V, corresponding to the retrovirus gag and pol genes, are separated by a short intergenic region, suggesting that in this virus and in contrast to retroviruses, fusion of gag and pol gene products is not obligatory.
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PLEASE ENTER A COMMAND OR BE LOGGED OFF IN 5 MINUTES
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28mar01 11:14:41 User208669 Session D1813.2
\$3.37 0.602 DialUnits File5
\$0.00 21 Type(s) in Format 6
\$9.90 6 Type(s) in Format 7
\$9.90 27 Types
\$13.27 Estimated cost File5
\$0.45 TYMNET
\$13.72 Estimated cost this search
\$13.94 Estimated total session cost 0.661 DialUnits
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28mar01 14:04:36 User208669 Session D1814.1
\$0.20 0.059 DialUnits File1
\$0.20 Estimated cost File1
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File 5:Biosis Previews(R) 1969-2001/Mar W3
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S2 219 S1 AND (POLYMERASE OR POL OR REV OR TRANSCRIPTASE)
S3 0 VARIANT? AND S2
S4 6 SUBSTIT? AND S2
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DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.
11481344 BIOSIS NO.: 199800262676
Variability in coat protein sequence homology among American and European sources of strawberry vein banding virus.
AUTHOR: Mraž Ivan(a); Petřík K; Šip M; Franová-Honečnárova J
AUTHOR ADDRESS: (a)Dep. Plant Virol., Inst. Plant Mol. Biol., Acad. Sci. Czech Republic, Branišovská 31, 370 05 České Budějovice, Czech Republic
JOURNAL: Plant Disease 82 (5):p544-546 May, 1998
ISSN: 0191-2917
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English
ABSTRACT: Strawberry vein banding virus (SVBV) isolates from North America, Czech Republic, Norway, and Germany were collected and their variability was determined by dot blot hybridization and confirmed by sequencing of a 431-nucleotide fragment from the middle part of the coat protein gene. Two different substitutions were found between the American and two Czech SVBV isolates, but the other isolates were identical in the compared region to the American isolate. Digoxigenin-labeled probes were prepared from these isolates and used for hybridization with polymerase chain reaction-amplified fragments of 26 Czech SVBV field isolates. No significant differences in the hybridization signal were found with any combination of samples and probes. These results show that the European isolates probably originate from a common ancestor and may have been introduced to Europe from America with planting or breeding material.

4/7/3
DIALOG(R)File 5:Biosis Previews(R)
(c) 2001 BIOSIS. All rts. reserv.
09996316 BIOSIS NO.: 199598451234
Gene I mutants of peanut chlorotic streak virus, a caulimovirus, replicate in plants but do not move from cell to cell.
AUTHOR: Ducasse D A; Mushegian A R; Shepherd R J(a)
AUTHOR ADDRESS: (a)Dep. Plant Pathol., Univ. Kentucky, Lexington, KY 40546
**USA

JOURNAL: Journal of Virology 69 (9):p5781-5786 1995
ISSN: 0022-538X
DOCUMENT TYPE: Article
RECORD TYPE: Abstract

Set Items Description
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S1 1826 CAULIMO? OR CAULIFLOWER(W)MOSAIC?

LANGUAGE: English

ABSTRACT: Gene I of peanut chlorotic streak virus (PCISV), a caulimovirus, is homologous to gene I of other caulimoviruses and may encode a protein for virus movement. To evaluate the function of gene I, several mutations were created in this gene of an infectious, partially redundant clone of PCISV. Constructs with an in-frame deletion and a single amino acid substitution in gene I were not infectious. To test for replication of these mutants in primarily infected cells, an immunosorbent PCR technique was devised. Virus particles formed by mutants in plants were recovered by binding to antivirus antibodies on a solid matrix and DNase treated to discriminate against residual inoculum, and DNA of trapped virions was subjected to PCR amplification. Gene I mutants were shown to direct formation of encapsulated DNA as revealed by a PCR product. Control gene V mutants (reverse transcriptase essential for replication) did not yield a PCR product. Quantitative PCR allowed estimation of the proportion of cells initially infected by gene I mutants and the amount of extractable virus per cell. It is concluded that PCISV gene I encodes a movement protein and that the immunoselection PCR technique is useful in studying subliminal virus infection in plants.

4/7/5

DIALOG(R)File 5.Biosis Previews(R)

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09304020 BIOSIS NO.: 199497312390

Patterns of nucleotide sequence variation among cauliflower mosaic virus isolates.

AUTHOR: Chenault K D; Melcher U (a)

AUTHOR ADDRESS: (a)Dep. Biochem. and Molecular Biol., Okla. State Univ., Stillwater, OK 74078-0454**USA

JOURNAL: Biochimie (Paris) 76 (1):p3-8 1994

ISSN: 0300-9084

DOCUMENT TYPE: Article

RECORD TYPE: Abstract

LANGUAGE: English

ABSTRACT: A consensus nucleotide sequence of the DNA of nine isolates of cauliflower mosaic virus (CaMV) was used to examine variation of nucleotide sequence in CaMV. Variability in coding regions was lowest in open reading frames (ORFs) 1, 2, 3 and 5 and higher in ORFs 4 and 6. Silent substitutions were not uniformly distributed among the ORFs. The large intergenic region was also variable, particularly in loops and bulges of a predicted secondary structure for this region of the 35S RNA transcript. A profile of frequencies of the substitution of consensus nucleotides with other nucleotides revealed a deficit of A to G transitions and an excess of transversions involving A. Most insertions/deletions could be accounted for by template misalignment during replication. The results suggest that the major source of variation in CaMV DNA sequences is associated with replication by reverse

transcription.

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